degree to which the temperature was lowered, and to the length of time during which the low temperature was maintained.

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Laking of blood by drying.

By **C. C. GUTHRIE** and **M. E. LEE** (by invitation).

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Drying of blood may cause laking of the scarlet blood discs.[1] To obtain more information regarding the phenomenon, the expeiments reported in this communication were undertaken.

The phenomenon may be produced in a number of ways. Perhaps the simplest is to prepare an ordinary wet blood mount and to observe it with a microscope. Another method is to make an ordinary blood smear and allow it to dry quickly in the air and then place it film side down on a microscope slide and after focusing it under a microscope, place a drop of serum, salt solution or other liquid on the slide, so that the edge of the drop forms contact with the edge of the slip and will spread under it.

Under these conditions laking occurs and may be readily observed in the individual discs.

In the case of a wet mount, the process is slow and various stages may be seen. If drying be rapid the individual discs may lose their hemoglobin with slight or no change in size. But if it be more gradual, they may be seen to swell before laking. This is also the case with shrunken or crenated discs.

In the case of dry mounts, the hemoglobin is almost instantaneously dissolved on contact with the liquid under the slip. If any change in the size of the discs occurs, it has not been observed.

At present the manner in which laking is thus produced is not known. Drying is, of course, accompanied by concentration of salts or other substances present in solution or suspension both within the discs and in the serum. Now it is known that hypertonic solutions of such substances if caused to act on the blood may produce laking. Therefore, the question arises, Is laking

[1] *Am. Jr. Phy.*, 1903, VIII, 441.

through drying the same fundamentally as laking by hypertonic solutions? Brahmachari[1] suggests that hypertonic solutions of sodium chloride lake by uniting with some cellular constituent and thus alter its normal properties. If this is true, it would seem that such union is at least of a doubtful chemical character; for a number of inorganic salts as well as cane sugar and glycerine in hypertonic solution may cause laking.

It is conceivable that the abstraction of sufficient water from the discs in any manner as by evaporation or by hypertonic solutions or by freezing, may so alter the molecular arrangement of the essential structures that it is not possible for them subsequently to imbibe or otherwise take up water and regain their normal properties,—hence laking.

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The schizogony in the life-cycle of Sarcocystis muris.

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The first period of the life cycle of *Sarcocystis muris* extends from the date of infection to the entrance of the unicellular parasite into the muscular tissue of the host (20 to 30 days).[2] In my former publications I could only describe from this period some large ameboid forms found in the walls of the intestine and in the lymph vessels of experimentally infected mice.[3]

My present investigation shows the appearance of small ameboid and schizogony forms six days after infection. These stages were discovered after feeding sarcosporidia to young mice nourished with milk from birth to the end of the experiments. These small schizogony forms (0.3 to 0.4 μ) consist of a tiny protoplasmic body with a caryosome-nucleus, and arise from smaller ameboid organisms which show typical schizogony.

[1] *Bio-Chem. Jr.*, 1909, IV, 59.

[2] Erdmann, "Die Entwicklung der *Sarcocystis muris* in der Muskulatur," *Sitzungsberichte der Gesellschaft Naturforschender Freunde*, 1910, p. 399.

[3] Ibidem, p. 382. Also cf. Erdmann, "Beiträge zur Morphologie und Entwickelungsgeschichte des Hammelsarkosporids in der Maus," *Centrb. für Bakt. und Parasitk.*, 1910, Bd. 53, Abt. I Orig., p. 515.